PB89-119911

Health and Environmental Effects Profile for Benzo(K)Fluoranthene

(U.S.) Environmental Protection Agency
Cincinnati, OH

Aug 87

U.S. Department of Commerce
National Technical Information Service
NTIS

TECHNICAL REPORT DATA
(Please read Instructions on the reverse before completing)

1. REPORT NO. EPA/600/X-87/394	2.
3. RECIPIENT'S ACCESSION NO	
4. TITLE AND SUBTITLE Health and Environmental Effects Profile for Benzo(k)fluoranthene	5. REPORT DATE August 1988
	6. PERFORMING ORGANIZATION CODE
7. AUTHOR(S)	8. PERFORMING ORGANIZATION REPORT NO
9. PERFORMING ORGANIZATION NAME AND ADDRESS	10. PROGRAM ELEMENT NO.
	11. CONTRACT/GRANT NO
12. SPONSORING AGENCY NAME AND ADDRESS Environmental Criteria and Assessment Office Office of Research and Development U.S. Environmental Protection Agency Cincinnati, OH 45268	13. TYPE OF REPORT AND PERIOD COVERED
	14. SPONSORING AGENCY CODE EPA/600/22
15. SUPPLEMENTARY NOTES	

16. ABSTRACT

The Health and Environmental Effects Profile for benzo(k)fluroanthene was prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste and Emergency Response to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA) and to provide health-related limits for emergency actions under Section 101 of the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA). Both published literature and information obtained from Agency program office files were evaluated as they pertained to potential human health, aquatic life and environmental effects of hazardous waste constituents.

Quantitative estimates have been presented provided sufficient data are available. Existing data are insufficient to determine a Reference Dose (RfD) or a carcinogenic potency factor for benzo(k)fluoranthene.

The Reportable Quantity (RQ) value of 1, 10, 100, 1000 or 5000 pounds is used to determine the quantity of a hazardous substance for which notification is required in the event of a release as specified by CERCLA based on chronic toxicity. Existing data are insufficient to determine an RQ value.

17. KEY WORDS AND DOCUMENT ANALYSIS

a. DESCRIPTORS	b. IDENTIFIERS/OPEN ENDED TERMS	c. COSATI Field Group

18. DISTRIBUTION STATEMENT Public	19. SECURITY CLASS (This Report) Unclassified	21. NO. OF PAGES 60
	20. SECURITY CLASS (This page) Unclassified	22. PRICE A04 $15.95

EPA Form 2220-1 (Rev. 4-77) PREVIOUS EDITION IS OBSOLETE

PB89-119911

EPA/600/X-87/394
August 1987

HEALTH AND ENVIRONMENTAL EFFECTS PROFILE FOR BENZO(K)FLUORANTHENE

ENVIRONMENTAL CRITERIA AND ASSESSMENT OFFICE
OFFICE OF HEALTH AND ENVIRONMENTAL ASSESSMENT
OFFICE OF RESEARCH AND DEVELOPMENT
U.S. ENVIRONMENTAL PROTECTION AGENCY
CINCINNATI, OH 45268

REPRODUCED BY
U.S. DEPARTMENT OF COMMERCE
NATIONAL TECHNICAL INFORMATION SERVICE
SPRINGFIELD, VA. 22161

DISCLAIMER

This document has been reviewed in accordance with U.S. Environmental Protection Agency policy and approved for publication. Mention of trade names or commercial products does not constitute endorsement or recommendation for use.

PREFACE

Health and Environmental Effects Profiles (HEEPs) are prepared for the Office of Solid Waste and Emergency Response by the Office of Health and Environmental Assessment. The HEEPs are intended to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA), as well as to provide health-related limits for emergency actions under Section 101 of the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA). Both published literature and information obtained from Agency program office files are evaluated as they pertain to potential human health, aquatic life and environmental effects of hazardous waste constituents. The literature searched and the dates of the searches are included in the section titled "Appendix: Literature Searched." The literature search material is current through November, 1985.

Quantitative estimates are presented provided sufficient data are available. For systemic toxicants, these include Reference doses (RfDs) for chronic exposures. An RfD is defined as the amount of a chemical to which humans can be exposed on a daily basis over an extended period of time (usually a lifetime) without suffering a deleterious effect. In the case of suspected carcinogens, RfDs are not estimated in this document series. Instead, a carcinogenic potency factor of q_1^* is provided. These potency estimates are derived for both oral and inhalation exposures where possible. In addition, unit risk estimates for air and drinking water are presented based on inhalation and oral data, respectively.

Reportable quantities (RQs) based on both chronic toxicity and carcinogenicity are derived. The RQ is used to determine the quantity of a hazardous substance for which notification is required in the event of a release as specified under CERCLA. These two RQs (chronic toxicity and carcinogenicity) represent two of six scores developed (the remaining four reflect ignitability, reactivity, aquatic toxicity and acute mammalian toxicity).

The first draft of this document was prepared by Syracuse Research Corporation under EPA Contract No. 68-03-3228. The document was subsequently revised after reviews by staff within the Office of Health and Environmental Assessment: Carcinogen Assessment Group, Reproductive Effects Assessment Group, Exposure Assessment Group, and the Environmental Criteria and Assessment Office in Cincinnati.

The HEEPs will become part of the EPA RCRA and CERCLA dockets.

ACKNOWLEDGEMENTS

The initial draft of this report was prepared by Syracuse Research Corporation under Contract No. 68-03-3228 for U.S. EPA's Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH. Dr. Christopher DeRosa was the Technical Project Monitor and Helen Ball was the Project Officer. The final documents in this series were prepared for the Office of Solid Waste, Washington, DC.

Scientists from the following U.S. EPA offices provided review comments for this document series:

Environmental Criteria and Assessment Office, Cincinnati, OH
Carcinogen Assessment Group
Exposure Assessment Group
Reproductive Effects Assessment Group

Editorial review for the document series was provided by:
Judith Olsen and Erma Durden
Environmental Criteria and Assessment Office, Cincinnati, OH

Technical support services for this document series were provided by:
Bette Zwayer, Pat Daunt, Karen Mann, and Jacky Bohanon
Environmental Criteria and Assessment Office, Cincinnati, OH.

EXECUTIVE SUMMARY

Benzo[k]fluoranthene is a pale yellow solid at ambient temperatures. It is soluble in ethanol, benzene and acetic acid, but is practically insoluble in water (Weast, 1980; Pearlman et al., 1984). This compound is susceptible to oxidation by ozone, peroxides and other oxidants. Frequently, dione is the product of such oxidation, although dimerization may occur in some cases (NAS, 1972). Benzo[k]fluoranthene is neither commercially produced nor used in the United States (IARC, 1983).

If released to the aquatic environment, benzo[k]fluoranthene is not expected to hydrolyze, oxidize (by RO_2 radical or $^1O_2$) or volatilize significantly (Mabey et al., 1981; Lyman et al., 1982). A static culture flask-screening biodegradation study with domestic wastewater seed has shown that benzo[k]fluoranthene can be significantly biodegraded (Fochtman, 1981); however, microbial oxidation of PAH requires oxygen and will not proceed in anoxic sediments or water (U.S. EPA, 1986a). In natural water, biodegradation is expected to be slow. In the dissolved state, direct photolysis may be significant in the water column; however, photolysis will be insignificant in deep, turbid waters. Adsorption to suspended particulate matters and sediments in water is an important environmental fate process (U.S. EPA, 1986a). The very low water solubility and high log K_{ow} of benzo[k]fluoranthene suggest a significant potential for bioaccumulation; however, PAH may not appreciably bioconcentrate in organisms such as fish that have microsomal oxidase because this metabolizes PAH (Santodonato et al., 1981). Therefore, bioaccumulation potential may be dependent on the organism being considered. The nonvariability of benzo[k]fluoranthene concentrations in bottom sediment cores in remote lakes (Tan and Heit, 1981) indicate that benzo[k]fluoranthene is very persistent under anaerobic and dark conditions.

If released to the atmosphere, benzo[k]fluoranthene will exist primarily in the particulate (adsorbed) phase, although vapor phase benzo[k]fluoranthene may also be present. Vapor phase benzo[k]fluoranthene appears susceptible to significant transformation by direct photolysis and reaction with ozone and HO radical (Lane and Katz, 1977; U.S. EPA, 1986b). Particulate phase benzo[k]fluoranthene may be more resistant to reactions as indicated by long distance transport of the atmospheric aerosol (Tan and Heit, 1981; Lunde and Bjoerseth, 1977). This compound is expected to be physically removed from the atmosphere by wet and dry deposition (Ligocki et al., 1985a; Tan and Heit, 1981). If released to soil, benzo[k]fluoranthene may be susceptible to slow biodegradation under aerobic conditions. Under most conditions, it is not expected to leach or volatilize and may persist in soils.

Human exposure to benzo[k]fluoranthene occurs primarily through the inhalation of tobacco smoke and polluted air and by the ingestion of contaminated food and water (IARC, 1983). The U.S. EPA (1982) reported that ~96% (2700 kkg) PAH are emitted to the atmosphere; of these the total release of benzo[k]fluoranthene comprises ~210 kg/year. The compound occurs ubiquitously as a product of incomplete combustion and naturally in fossil fuels (IARC, 1983). It has been widely detected in drinking water, surface water, groundwater, rainwater and aquatic sediments (see Tables 3-1 and 3-2), in many foods (Dennis et al., 1983), and in the ambient atmosphere (see Table 3-3). The presence of benzo[k]fluoranthene in food is a result of contamination from a polluted environment and formation during the cooking process (Santodonato et al., 1981; Fazio and Howard, 1983). The average dietary intake of benzo[k]fluoranthene in England has been estimated to be 0.06 µg/day (Dennis et al, 1983). The average intake of this compound from drinking water in the United States has been estimated to be 0.2 ng/day,

while the inhalation intake in the United States has been estimated to be 0.6-20 ng/day. Its concentration in the ambient atmosphere has apparently been decreasing over the past 25 years (Santodonato et al., 1981; Gordon and Bryan, 1973). The higher atmospheric levels of this compound in wintertime compared with summertime may be due to increased use of fossil fuel combustion for heating purposes (Greenberg et al., 1985).

Data concerning the toxicity of benzo[k]fluoranthene to aquatic organisms could not be located in the available literature as cited in the Appendix. The only information consisted of monitoring data for various species and locations. Residues in species that are commonly eaten by humans were mussels in Norway, 6-69 ng/g (Knutzen and Sortland, 1982), clams in Japan, 0.12-0.92 ng/g (Tsuji et al., 1985), and lobster from Eastern Canada, 0.35 ng/g before impoundment and 169 ng/g after impoundment (Dunn and Fee, 1979).

Information regarding the absorption, distribution or excretion of benzo[k]fluoranthene could not be located in the available literature as cited in the Appendix.

In vitro studies with rat liver preparations have shown that 8,9-dihydro-8,9-dihydroxybenzo[k]fluoranthene is the major metabolite of benzo[k]-fluoranthene (LaVoie et al., 1980; Hecht et al., 1980).

Single intrapulmonary injections of 0.16, 0.83 or 4.15 mg benzo[k]fluoranthene (99.5% pure) in beeswax trioctanoin mixture into groups of 27-35 rats produced dose-related squamous cell carcinomas of the lung after lifetime observation (Deutsch-Wenzel et al., 1983). Tumors were not observed in groups of 35 vehicle or untreated controls, and incidences in the low-, middle- and high-dose treated groups were 0/35, 3/31 and 12/27, respectively.

The carcinogenicity of benzo[k]fluoranthene has also been evaluated in dermal studies with mice involving 2 or 3 times weekly applications for life or 13 months (Wynder and Hoffman, 1959; Habis et al., 1980), in mouse-skin initiation-promotion assays using TPA as a promoter (LaVoie et al., 1982; Amin et al., 1985), and in a subcutaneous injection study in which mice were given three injections at monthly intervals (Lacassagne et al., 1963). Benzo[k]fluoranthene was active as an initiator in the initiation-promotion assays and produced injection site sarcomas in the subcutaneous study. Interpretation of the subcutaneous injection study is complicated, however, by the lack of vehicle or untreated controls and by an unspecified observation period.

Benzo[k]fluoranthene induced mutations in Salmonella typhimurium strains TA100 and TA98 in the presence of exogenous metabolic activation preparations (LaVoie et al., 1980; Hermann et al., 1980; Amin et al., 1985).

Information regarding chronic or subchronic toxic effects, teratogenicity or other reproductive effects of benzo[k]fluoranthene could not be located in the available literature as cited in the Appendix.

There was sufficient evidence that benzo[k]fluoranthene is carcinogenic to animals, but the lack of studies by a relevant route precluded derivation of a q_1^* based on data specific for benzo[k]fluoranthene. According to IARC (1983), benzo[k]fluoranthene is a Group 2B chemical, meaning that it is probably carcinogenic to humans. The corresponding EPA classification is B2. Data were insufficient to derive an RQ based on chronic toxicity.

TABLE OF CONTENTS

		Page
1.	INTRODUCTION	1
1.1.	STRUCTURE AND CAS NUMBER	1
1.2.	PHYSICAL AND CHEMICAL PROPERTIES	1
1.3.	PRODUCTION DATA	2
1.4.	USE DATA	2
1.5.	SUMMARY	2
2.	ENVIRONMENTAL FATE AND TRANSPORT PROCESSES	3
2.1.	WATER	3
2.1.1.	Hydrolysis	3
2.1.2.	Oxidation	3
2.1.3.	Photolysis	3
2.1.4.	Microbial Degradation	3
2.1.5.	Volatilization	4
2.1.6.	Adsorption	4
2.1.7.	Bioconcentration	4
2.1.8.	Persistence	5
2.2.	AIR	5
2.2.1.	Degradation	6
2.2.2.	Physical Removal	7
2.3.	SOIL	7
2.3.1.	Microbial Degradation	7
2.3.2.	Chemical Degradation	7
2.3.3.	Adsorption	7
2.3.4.	Volatilization	8
2.3.5.	Persistence	8
2.4.	SUMMARY	8
3.	EXPOSURE	10
3.1.	WATER	10
3.2.	FOOD	14
3.3.	INHALATION	15
3.4.	DERMAL	17
3.5.	SUMMARY	17
4.	PHARMACOKINETCS	18
4.1.	ABSORPTION	18
4.2.	DISTRIBUTION	18
4.3.	METABOLISM	18
4.4.	EXCRETION	18
4.5.	SUMMARY	18

TABLE OF CONTENTS (cont.)

		Page
5.	EFFECTS	20
5.1.	CARCINOGENICITY	20
5.2.	MUTAGENICITY	21
5.3.	TERATOGENICITY	21
5.4.	OTHER REPRODUCTIVE EFFECTS	21
5.5.	CHRONIC AND SUBCHRONIC TOXICITY	21
5.6.	OTHER RELEVANT INFORMATION	24
5.7.	SUMMARY	24
6.	AQUATIC TOXICITY	25
6.1.	ACUTE	25
6.2.	CHRONIC	25
6.3.	PLANTS	25
6.4.	RESIDUES	25
6.5.	SUMMARY	27
7.	EXISTING GUIDELINES AND STANDARDS	28
7.1.	HUMAN	28
7.2.	AQUATIC	29
8.	RISK ASSESSMENT	30
9.	REPORTABLE QUANTITIES	31
9.1.	REPORTABLE QUANTITY (RQ) RANKING BASED ON CHRONIC TOXICITY	31
9.2.	WEIGHT OF EVIDENCE AND POTENCY FACTOR ($F=1/ED_{10}$) FOR CARCINOGENICITY	31
10.	REFERENCES	34
APPENDIX:	LITERATURE SEARCHED	45

LIST OF TABLES

No.	Title	Page
3-1	Benzo[k]fluoranthene Monitoring Data for Various Types of Water.	11
3-2	U.S. Sediment Monitoring Data for Benzo[k]fluoranthene. . . .	13
3-3	U.S. Air Monitoring Data for Benzo[k]fluoranthene During 1979-1983	16
5-1	Dermal Injection Carcinogenicity Studies of Benzo[k]fluoranthene.	22
5-2	Mutagenicity Testing of Benzo[k]fluoranthene.	23
6-1	Monitoring Data for Benzo[k]fluoranthene in Marine Species from Norwegian Waters	26
9-1	Benzo[k]fluoranthene: Minimum Effective Dose (MED) and Reportable Quantity (RQ).	32

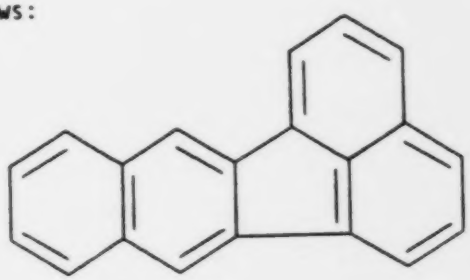
LIST OF ABBREVIATIONS

BCF	Bioconcentration factor
CAS	Chemical Abstracts Service
K_{oc}	Soil sorption coefficient
K_{ow}	Octanol/water partition coefficient
PAH	Polycyclic aromatic hydrocarbons
ppb	Parts per billion
ppm	Parts per million
ppt	Parts per trillion
TLV	Threshold limit value
TPA	Terephthalic acid
TWA	Time-weighted average
UV	Ultraviolet

1. INTRODUCTION

1.1. STRUCTURE AND CAS NUMBER

The chemical commonly called benzo[k]fluoranthene is also known as 11,12-benzofluoranthene; 8,9-benzfluoranthene; B[k]F; and 11,12-benzo[k]-fluoranthene (U.S. EPA, 1986a; Santodonato et al., 1981). The structure, empirical formula, molecular weight and CAS Registry number for this chemical are as follows:



Empirical formula: $C_{20}H_{12}$

Molecular weight: 252.32

CAS Registry number: 207-08-9

1.2. PHYSICAL AND CHEMICAL PROPERTIES

Benzo[k]fluoranthene is a pale yellow crystalline solid at ambient temperatures. It is soluble in ethanol, benzene and acetic acid, but practically insoluble in water (Weast, 1980). Some of the physical properties of this compound are listed below:

Melting point:	217°C	Weast, 1980
Boiling point:	480°C	Weast, 1980
Water solubility at 25°C:	0.76 µg/ℓ	Pearlman et al., 1984
Log K_{ow}:	6.04-6.44 (estimated)	Readman et al., 1982; Ruepert et al., 1985
Vapor pressure at 25°C:	9.59×10^{-11} mm Hg	Santodonato et al., 1981

Henry's Law constant:	4.19×10^{-8} atmos-m^3/mol (estimated)

Since this compound is a PAH, it is expected to be reasonably chemically reactive. It can undergo substitution and addition reaction, and is susceptible to oxidation by ozone, peroxides and other oxidants (NAS, 1972). Frequently, dione is the product of such oxidation, although dimerization may occur in some cases (NAS, 1972).

1.3. PRODUCTION DATA

Benzo[k]fluoranthene is neither commercially produced nor imported into the United States (IARC, 1983; USITC, 1984).

1.4. USE DATA

There is no known commercial use of benzo[k]fluoranthene (IARC, 1983). Small amounts of this compound are used for scientific research.

1.5. SUMMARY

Benzo[k]fluoranthene is a pale yellow solid at ambient temperatures. It is soluble in ethanol, benzene and acetic acid, but is practically insoluble in water (Weast, 1980; Pearlman et al., 1984). This compound is susceptible to oxidation by ozone, peroxides and other oxidants. Frequently, dione is the product of such oxidation, although dimerization may occur in some cases (NAS, 1972). Benzo[k]fluoranthene is neither commercially produced nor used in the United States (IARC, 1983).

2. ENVIRONMENTAL FATE AND TRANSPORT PROCESSES

2.1. WATER

2.1.1. Hydrolysis. Benzo[k]fluoranthene contains no hydrolyzable functional groups; therefore, hydrolysis is not expected to be significant (Mabey et al., 1981).

2.1.2. Oxidation. The rate constants for the oxidation of benzo[k]fluoranthene with photochemically produced RO_2 radical and $^1O_2$ have been estimated to be 5×10^3 and 4×10^7 M^{-1} $hour^{-1}$, respectively, at 25°C (Mabey et al., 1981). Assuming the RO_2 radical and $^1O_2$ concentrations of natural waters are 10^{-9} and 10^{-12} M (Mabey et al., 1981), respectively, the respective half-lives are estimated to be 15.8 and 2.0 years. Therefore, these reactions are not environmentally significant.

2.1.3. Photolysis. In a cyclohexane solvent, benzo[k]fluoranthene exhibits UV absorption maxima at 295, 306, 358, 370, 378 and 400 nm (IARC, 1983); therefore, direct photolysis in sunlight is a possibility. Muel and Saguem (1985) exposed a 28.6 µg/ℓ n-heptane solution of benzo[k]fluoranthene to 1 month of November sunlight and found that 72% had degraded after the exposure period. This would suggest that direct photolysis in the dissolved state in the water column is possible.

The majority of benzo[k]fluoranthene will be present, however, in the particle-sorbed state in water (Section 2.1.6.) and may not readily photolyze. Therefore, photolysis may be relatively more significant in shallow, clear water than in deep, turbid water. Overall, photodegradation may not be as important as other processes in water.

2.1.4. Microbial Degradation. Fochtman (1981) examined the biodegradability of benzo[k]fluoranthene in a static culture flask-screening procedure in which 1% of the compound was dissolved in an emulsifier and added to

the bacterial suspension (domestic wastewater seed) at a concentration of 1-2 ppm. The suspension was incubated for 7 days. A fresh bacterial suspension was prepared weekly using the seed from the previous week and the procedure was continued for 28 days. Approximately 54% of the benzo[k]-fluoranthene was typically degraded during a 7-day incubation.

In natural waters, PAH with four or more aromatic rings are degraded slowly by microbes, and biodegradation is considered to be the ultimate fate process (U.S. EPA, 1986a); however, the concentrations of microorganisms capable of oxidizing the hydrocarbons are extremely low in all but heavily polluted fresh and marine waters, and most species of microorganisms cannot use PAH as a sole carbon source. Microbial oxidation of PAH requires oxygen and will not proceed in anoxic sediments or water (U.S. EPA, 1986a).

2.1.5. Volatilization. Based on a water solubility of 0.00076 ppm at 25°C (Pearlman et al., 1984) and a vapor pressure of 9.59×10^{-11} mm Hg at 25°C (Santodonato et al., 1981), Henry's Law constant for benzo[k]fluoranthene can be estimated to be 4.2×10^{-8} atm-m³/mol. This value of Henry's Law constant indicates that benzo[k]fluoranthene is not expected to volatilize significantly from the aquatic environment (Lyman et al., 1982).

2.1.6. Adsorption. The estimated K_{oc} value of benzo[k]fluoranthene is nearly 1 million (Section 2.3.3.) and the widespread detection of benzo[k]-fluoranthene in various sediments (Section 3.1.) indicates that adsorption to suspended particulate matters and sediments is an important environmental fate process. Movement by sediments is considered to be an important transport process for PAH (U.S. EPA, 1986a).

2.1.7. Bioconcentration. Estimation of BCF can be made from the following equations (Lyman et al., 1982):

$$\log BCF = 0.76 \log K_{ow} - 0.23 \qquad (2\text{-}1)$$

$$\log BCF = 2.791 - 0.564 \log \text{water solubility (in ppm)} \qquad (2\text{-}2)$$

Based on a water solubility of 0.00076 ppm at 25°C (Pearlman et al., 1984) and a log K_{ow} of 6.12 (U.S. EPA, 1986b), the BCF values estimated from Equations 2-1 and 2-2 for benzo[k]fluoranthene are 35,500 and 26,400, respectively, which suggest significant bioaccumulation potential. PAH, however, may not appreciably bioconcentrate in organisms such as fish that have microsomal oxidase because this enzyme metabolizes PAH (Santodonato et al., 1981). Therefore, the bioaccumulation potential may be dependent on the organism being considered.

2.1.8. Persistence. Tan and Heit (1981) monitored sediment cores taken from Woods Lake in the remote Adirondack Forest of upstate New York for various PAH. The following benzo[k]fluoranthene concentrations (ng/g dry sediment) were found at various depths: 560 (0-4 cm), 180 (4-8 cm), 11 (8-11 cm), 4 (12-17 cm), 7 (24-26 cm), 5 (42-44 cm) and 8 (80-84 cm). The constancy in the concentration of benzo[k]fluoranthene in the deeper sediment cores indicates that the compound is very persistent under anaerobic and dark conditions present in the deep sediment cores.

2.2. AIR

Benzo[k]fluoranthene exists in the ambient atmosphere predominantly in particulate-associated form. From their ambient air monitoring data collected in Portland, OR, Ligocki et al. (1985a,b) found the combined mean benzo[b,j,k]fluoranthene concentration in the vapor phase was 0.11 ng/m³, while the combined mean concentration of the same three compounds in the particulate phase was 3.4 ng/m³. Yamasaki et al. (1985) conducted a laboratory experiment in which ambient airborne particulates were exposed to PAH-free air for 48 hours at 9.1°C and found that ~95% of the benzo[k]-fluoranthene remained adsorbed to the particulates. Cautreels and Van Cauwenberghe (1978) found that the concentration of benzo[k]fluroanthene in

the particulate phase was ~12 times higher than its concentration in the gas phase. In a monitoring study of the air over the tropical and equatorial eastern Atlantic Ocean, Marty et al. (1984) detected benzo[k]fluoranthene in the particulates but not in the vapor phase. The form in which benzo[k]-fluoranthene exists in the atmosphere has a significant bearing on its environmental fate. In general, the compound will be less reactive in the particulate phase than in the gas phase.

2.2.1. Degradation. Lane and Katz (1977) used simulated atmospheric conditions to expose pure benzo[k]fluoranthene to ozone, a Quartzline lamp with spectral distribution very close to solar distribution between 295 and 400 nm, or a combination of both light and ozone. The half-life using UV light only was 14.1 hours, while the half-life with ozone (0.19 ppm) in the dark was 34.9 hours; a combination of both light and ozone resulted in a half-life of 3.9 hours. These results suggest that a combination of photolysis in the presence of ozone may induce faster transformation of benzo[k]-fluoranthene than either of the processes alone. It is, however, difficult to assess the significance of this reaction in the ambient atmosphere because the concentrations of O_3 used in these experiments were higher than normally found in the atmosphere.

The half-life for the vapor phase reaction of benzo[k]fluoranthene with photochemically produced HO radical has been estimated to be ~1 day at 25°C assuming an average atmospheric HO radical concentration of 8×10^5 molecules/cm^3 (U.S. EPA, 1986b).

Data specific to the degradation of particulate adsorbed benzo[k]fluor-anthene could not be located in the available literature as cited in the Appendix. Behymer and Hites (1985) examined the atmospheric photodegrada-tion of 15 other PAHs that were adsorbed to various substrates (silica gel,

alumina, fly ash and carbon black) by using a laboratory photoreactor to simulate atmospheric conditions. In general, significant photolysis was observed when the adsorbing substrate was either silica gel, alumina or fly ash; however, carbon black adsorption clearly stabilized the phototransformation of the PAH. In the atmosphere, adsorption to stabilizing substrates will allow PAH to be transported over long distances in the atmospheric aerosol because of increased persistence. Lunde and Bjoerseth (1977) reported that benzo[k]fluoranthene was transported in the atmospheric aerosol from England to Norway.

2.2.2. Physical Removal. Removal of adsorbed benzo[k]fluoranthene from the atmosphere may occur by wet and dry deposition. Particulate-associated benzo[k]fluoranthene has been detected in rainwater (Ligocki et al., 1985b). The presence of benzo[k]flouranthene in lake sediments in the Adirondack Forest in New York has been attributed to physical deposition (Tan and Heit, 1981). Dissolved benzo[k]fluoranthene has also been detected in rainwater (Ligocki et al., 1985a), suggesting that physical removal by washout or dissolution into clouds with subsequent rainfall may be possible.

2.3. SOIL

2.3.1. Microbial Degradation. Benzo[k]fluoranthene may possibly undergo slow biodegradation in soil under aerobic conditions (see Section 2.1.4.); however, additional experimental data are required to predict the rate at which biodegradation may occur.

2.3.2. Chemical Degradation. No data are available to indicate that benzo[k]fluoranthene is transformed chemically in natural soils.

2.3.3. Adsorption. Estimation of K_{oc} can be made from the following regression equations (Lyman et al., 1982):

$$\log K_{oc} = 0.44 - 0.54 \log \text{water solubility (in mol fraction)} \quad (2\text{-}3)$$

$$\log K_{oc} = 1.00 \log K_{ow} - 0.21 \quad (2\text{-}4)$$

Based on a water solubility of 0.76 µg/ℓ (Pearlman et al., 1984) at 25°C and a log K_{ow} of 6.12 (U.S. EPA, 1986b), the K_{oc} values estimated from Equations 2-3 and 2-4 for benzo[k]fluoranthene are 0.96 and 0.81 million, respectively, which suggest soil immobility. The detection of benzo[k]-fluoranthene in several groundwaters (Section 3.1.), however, indicates that leaching can occur. This leaching may occur under certain conditions, such as from soils with low organic content (e.g., sand) or high porosity, or from sites that have been exposed to spills or chemical wastes containing benzo[k]fluoranthene. Benzo[k]fluoranthene is not expected to leach in soil under most other conditions.

2.3.4. Volatilization. Volatilization of benzo[k]fluoranthene from soils is not expected to be an important process (Sims and Overcash, 1983).

2.3.5. Persistence. When seven applications of PAH-containing oil sludge was amended to a sandy loam soil over a 2-year period, and then monitored for an additional 1.5 years, 30% of the total benzo[k]fluoranthene application remained after the total 3.5 years (Bossert et al., 1984). Therefore, this compound is expected to persist in most soils.

2.4. SUMMARY

If released to the aquatic environment, benzo[k]fluoranthene is not expected to hydrolyze, oxidize (by RO_2 radical or $^1O_2$) or volatilize significantly (Mabey et al., 1981; Lyman et al., 1982). A static culture flask-screening biodegradation study with domestic wastewater seed has shown that benzo[k]fluoranthene can be significantly biodegraded (Fochtman, 1981); however, microbial oxidation of PAH requires oxygen and will not proceed in anoxic sediments or water (U.S. EPA, 1986a). In natural water, biodegradation is expected to be slow. In the dissolved state, direct photolysis may

be significant in the water column; however, photolysis will be insignificant in deep, turbid waters. Adsorption to suspended particulate matters and sediments in water is an important environmental fate process (U.S. EPA, 1986a). The very low water solubility and high log K_{ow} of benzo[k]fluoranthene suggest a significant potential for bioaccumulation; however, PAH may not appreciably bioconcentrate in organisms such as fish that have microsomal oxidase because this metabolizes PAH (Santodonato et al., 1981). Therefore, bioaccumulation potential may be dependent on the organism being considered. The nonvariability of benzo[k]fluoranthene concentrations in bottom sediment cores in remote lakes (Tan and Heit, 1981) indicate that benzo[k]fluoranthene is very persistent under anaerobic and dark conditions. If released to the atmosphere, benzo[k]fluoranthene will exist primarily in the particulate (adsorbed) phase, although vapor phase benzo[k]fluoranthene may also be present. Vapor phase benzo[k]fluoranthene appears susceptible to significant transformation by direct photolysis and reaction with ozone and HO radical (Lane and Katz, 1977; U.S. EPA, 1986b). Particulate phase benzo[k]fluoranthene may be more resistant to reactions as indicated by long distance transport of the atmospheric aerosol (Tan and Heit, 1981; Lunde and Bjoerseth, 1977). This compound is expected to be physically removed from the atmosphere by wet and dry deposition (Ligocki et al., 1985a; Tan and Heit, 1981). If released to soil, benzo[k]fluoranthene may be susceptible to slow biodegradation under aerobic conditions. Under most conditions, it is not expected to leach or volatilize and may persist in soils.

3. EXPOSURE

Human exposure to benzo[k]fluoranthene occurs primarily through the inhalation of tobacco smoke and polluted air and by the ingestion of contaminated food and water (IARC, 1983). The compound occurs ubiquitously as a product of incomplete combustion and occurs naturally in fossil fuels (IARC, 1983).

3.1. WATER

Table 3-1 lists various benzo[k]fluoranthene monitoring data for drinking water, groundwater, surface water and rainwater; Table 3-2 lists sediment monitoring data for various U.S. locations. In an analysis of the U.S. EPA STORET database, benzo[k]fluoranthene was detected in 1.8% of 1248 effluents and 3.0% of 873 surface waters (Staples et al., 1985). Griest (1980) detected benzo[b,] and k]fluoranthene at a concentration of 23.0 µg/g (dry sediment) in the sediment of an effluent channel from a coking plant. Benzo[k]fluoranthene was also identified in the effluent from a sewage treatment plant at levels <12 ppt (Kveseth et al., 1982). Shackelford and Keith (1976) detected benzo[k]fluoranthene in effluents from chemical plants, sewage treatment plants and in raw sewage. In the preliminary findings of the U.S. EPA Nationwide Urban Runoff Program, benzo[k]-fluoranthene was detected in the stormwater runoff from Bellevue, WA, and Lake Quinsigamond, MA, at levels of 4-10 ppb (Cole et al., 1984).

In general, PAH can be released to water from industrial and municipal treatment plant effluents, atmospheric fallout and precipitation, road runoff (tire wear, bitumen and asphalt surfaces, cracked lubricating oils) and marine shipping and harbor oil (Santodonato et al, 1981). Sorrell et

TABLE 3-1

Benzo[k]fluoranthene Monitoring Data for Various Types of Water

Type of Water	Concentration (ppt)	Location	Sampling Date	Reference
Drinking (finished)	0.4	Syracuse, NY	1976-1977	Saxena et al.,1977
	ND	Buffalo, NY	1977	
	0.2	Pittsburgh, PA	1977	
	0.2	Huntington, WV	1977	
	ND	Endicott, NY	1977	
	0.1	Hammondsport, NY	1977	
	ND	Philadelphia, PA	1977	
	0.7	New York, NY	1977	
	0.1	Lake George, NY	1977	
	0.6	New Orleans, LA	1977	
Drinking (finished)	<1.0	Cincinnati, OH	NR	Sorrell et al., 1980
	<1.0	New Orleans, LA	NR	
	<1.0	Miami, FL	NR	
	<1.0	Seattle, WA	NR	
	<1.0	Columbus, OH	NR	
	<1.0	Cape Girardeau, MO	NR	
	<1.0	Wheeling, WV	NR	
	<2.0	Jefferson Parish, LA	NR	
Drinking (tap)	0.02-0.10	Norway, Finland, Sweden	NR	Kveseth et al., 1982
	1-3.4	NR	NR	IARC, 1983
Groundwater	0.8-10.0	Germany	1963-1964	Sorrell et al., 1980
	3000 (maximum)	Netherlands	1976-1978	Zoeteman et al., 1981
	NR[a]	St. Louis Park, MN	post-1982	Rostad et al., 1985
	1-3.5	NR	NR	IARC, 1983

TABLE 3-1 (cont.)

Type of Water	Concentration (ppt)	Location	Sampling Date	Reference
Surface (lake)	NR[a]	Lakes Erie, Ontario, Michigan and Superior	NR	Great Lakes Water Quality Board, 1983
Surface (river)	0.1-1.0 (in solution)	River Trent (England)	NR	Sorrell et al., 1980
	0.5-265 (in suspended sediment)	River Trent (England)	NR	
	2.7-117.0	Germany	1963-1964	
	0.8-7.7	Severn River (England)	NR	
	4.2	Tamar Estuary (England)	1980	Readman et al., 1982
Surface	0.2-0.8	NR	NR	IARC, 1983
Rainwater	1.6 (mean)[b] (dissolved)	Portland, OR	1984	Ligocki et al., 1985a
	9.2 (average)[b] (particulate)	Portland, OR	1984	Ligocki et al., 1985b
	6-40	Netherlands	1982-1983	Van Noort and Wondergem, 1985

[a]Concentration not reported but the compound was qualitatively detected

[b]Benzo[b+j+k]fluoranthene

ND = Not detected; NR = not reported

TABLE 3-2

U.S. Sediment Monitoring Data for Benzo[k]fluoranthene

Concentration (ng/g)	Location	Sampling Date	Reference
12-26[a]	Washington State (river bottom sediment)	1979-1980	Prahl et al., 1984
8-140[a]	Columbia River (suspended sediment)	1979-1980	
88-250[b]	Cayuga Lake, NY (deepwater sediment)	1978	Heit, 1985
16-550[b]	Cayuga Lake, NY (littoral sediment)	1978	
2.9-86 (mean)[c]	Lake Pontchartrain, LA	1980	McFall et al., 1985
560 (0-4 cm depth)	Woods Lake, NY	NR	Tan and Heit, 1981
180 (4-8 cm depth)	Woods Lake, NY	NR	
8 (80-84 cm depth)	Woods Lake, NY	NR	
120 (0-4 cm depth)	Sagamore Lake, NY	NR	
110 (4-8 cm depth)	Sagamore Lake, NY	NR	
2 (71-75 cm depth)	Sagamore Lake, NY	NR	
14-696	Penobscot Bay, Gulf of Maine	1982	Johnson et al., 1985

[a]Benzo[j+k]fluoranthene

[b]Benzo[b+k]fluoranthene

[c]Unspecified isomers

NR = Not reported

al. (1980) suggested that benzo[k]fluoranthene and other PAHs may contaminate drinking water supplies as a result of coal tar or asphalt-based materials used to line transmission pipes and storage tanks.

Based on the monitoring data from Table 3-1, the concentration of benzo[k]fluoranthene in drinking water may be on the order of 0.1 ppt (ng/ℓ). Assuming an average daily water intake of 2.0 ℓ for an adult, the intake of benzo[k]fluoranthene from drinking water is estimated to be 0.2 ng/day.

3.2. FOOD

In general, PAHs found in food are present as a result of contamination from a polluted environment or are formed during the cooking process (Santodonato et al., 1981; Fazio and Howard, 1983); minute amounts of the chemical may originate from geochemical or biosynthetic sources (Fazio and Howard, 1983).

Fazio and Howard (1983) reported the detection of benzo[k]fluorathene in oysters, very dark coffee (0-0.8 µg/kg) and cooked Japanese horse mackerel (0.2 µg/kg).

Dennis et al. (1983) examined total diet samples of food groups in England and found the following mean benzo[k]fluoranthene concentrations (in µg/kg) in the various food groups: cereals (0.08), meat (0.01), fish (0.04), oils and fats (0.32), fruit and sugar (0.02), vegetables (0.02-0.07), beverages (0.003) and milk (0.003). The total benzo[k]fluoranthene dietary load was estimated to be 0.06 µg/person/day (Dennis et al., 1983). Based on the monitoring of total diet market basket samples collected in the Netherlands, Vaessen et al. (1984) estimated the median intake of benzo[b+j+k]fluoranthene to be 0.1 µg/day.

3.3. INHALATION

Table 3-3 lists recent (1979-1983) U.S. ambient air monitoring data for benzo[k]fluoranthene; in general, the mean average concentration detected is <1.0 ng/m³. Ambient air monitoring data for benzo[k]fluoranthene from 1958 to the mid-1970s (Santodonato et al., 1981; Gordon and Bryan, 1973) suggest that the ambient atmospheric load of benzo[k]fluoranthene has been generally decreasing over the past 25 years, possibly because of decreases in coal consumption for residential heating and industrial uses, improved disposal methods of solid wastes, restrictions on open burning and improved efficiencies for stationary incineration and combustion operations and improvement of pollution control. The higher atmospheric levels of benzo-[k]fluoranthene in wintertime verus summertime air in New Jersey (see Table 3-3) may be a reflection of the increased use of fossil fuel combustion for heating purposes.

Assuming the approximate average ambient air concentrations of benzo[k]-fluoranthene in the United States range from 0.03-1.0 ng/m³ (see Table 3-3) and an individual air intake of 20 m³/day, the average intake is estimated to be 0.6-20 ng/day. Matsumoto and Kashimoto (1985) used Japanese monitoring data to estimate an average daily intake of 15 ng.

In general, ~96% (27000 kkg) PAH are emitted to the atmosphere. Of the atmospheric emissions combustion of fossil fuels (oil, coal), gasoline and diesel exhaust, open burning (agricultural burning, forest fires, structural fires, refuse burning), the burning of wood especially for residential heating (NRC, 1983) and municipal/industrial incineration comprise ~99% of the benzo[a]pyrene group PAHs. U.S. EPA (1982) reported an estimate of 210 kkg/year benzo[k]fluoranthene released to the environment.

TABLE 3-3

U.S. Air Monitoring Data for Benzo[k]fluoranthene During 1979-1983

Concentration (ng/m^3)	Location	Sampling Date	Reference
0.18 (average)*	Quillayute, WA	1979	Prahl et al., 1984
0.19 (average)*	Sequim, WA	1979	
0.41 (average)*	Seattle, WA	1979	
0.11 (mean gas-phase)	Portland, OR	1984	Ligocki et al., 1985a
3.4 (average particulate phase)*	Portland, OR	1984	Ligocki et al., 1985b
0.03-0.3 (particulate phase)	Columbia, SC	1982	Keller and Bidleman, 1984
0.03-0.20 (mean)	4 cities - New Jersey	Summer 1981	Harkov et al., 1984
0.28-0.97 (mean)	4 cities - New Jersey	Winter 1982	Greenberg et al., 1985
0.04-0.11 (mean)	4 cities - New Jersey	Summer 1982	
0.08-0.63 (mean)	4 cities - New Jersey	Winter 1983	

*Benzo[b+j+k]fluoranthenes

Benzo[k]fluoranthene was identified in mainstream cigarette smoke (0.7-12 ng/cigarette) and mainstream marijuana smoke (11 ng/cigarette) (IARC, 1983). The concentration of benzo[k]fluoranthene detected in various fly ash samples ranged from not detected to 23 ng/g (Elceman et al., 1981).

3.4. DERMAL

Pertinent dermal monitoring data could not be located in the available literature as cited in the Appendix.

3.5. SUMMARY

Human exposure to benzo[k]fluoranthene occurs primarily through the inhalation of tobacco smoke and polluted air and by the ingestion of contaminated food and water (IARC, 1983). The compound occurs ubiquitously as a product of incomplete combustion and naturally in fossil fuels (IARC, 1983). It has been widely detected in drinking water, surface water, groundwater, rainwater and aquatic sediments (see Tables 3-1 and 3-2), in many foods (Dennis et al., 1983), and in the ambient atmosphere (see Table 3-3). The presence of benzo[k]fluoranthene in food is a result of contamination from a polluted environment and formation during the cooking process (Santodonato et al., 1981; Fazio and Howard, 1983). The average dietary intake of benzo-[k]fluoranthene in England has been estimated to be 0.06 µg/day (Dennis et al., 1983). The average intake of this compound from drinking water in the United States has been estimated to be 0.2 ng/day, while the inhalation intake in the United States has been estimated to be 0.6-20 ng/day. Its concentration in the ambient atmosphere has apparently been decreasing over the past 25 years (Santodonato et al., 1981; Gordon and Bryan, 1973). The higher atmospheric levels of this compound in wintertime compared with summertime may be due to increased use of fossil fuel combustion for heating purposes (Greenberg et al., 1985).

4. PHARMACOKINETICS

4.1. ABSORPTION

Specific data regarding the gastrointestinal or pulmonary absorption of benzo[k]fluoranthene could not be located in the available literature as cited in the Appendix. Data from other structurally related PAH suggest, however, that benzo[k]fluoranthene is absorbed readily from the gastrointestinal tract (Rees et al., 1971) and lungs (Kotin et al., 1969; Vainio et al., 1976). In general, PAH are highly lipid soluble and can pass across epithelial membranes (U.S. EPA, 1980a).

4.2. DISTRIBUTION

Pertinent data regarding the distribution of benzo[k]fluoranthene could not be located in the available literature as cited in the Appendix.

4.3. METABOLISM

8,9-Dihydro-8,9-dihydroxy benzo[k]fluoranthene has been identified as the major metabolite of benzo[k]fluoranthene in <u>in vitro</u> metabolism studies with rat liver S-9 preparations (LaVoie et al., 1980; Hecht et al., 1980). Benzo[k]fluoranthene-8,9-epoxide was not isolated but considered to be the likely precursor of the 8,9-dihydrodiol. Indirect evidence (mutagenicity) suggests that the 8,9-dihydrodiol may form a dihydrodiol-epoxide.

4.4. EXCRETION

Pertinent data regarding the excretion of benzo[k]fluoranthene could not be located in the available literature as cited in the Appendix.

4.5. SUMMARY

Information regarding the absorption, distribution or excretion of benzo[k]fluoranthene could not be located in the available literature as cited in the Appendix.

In vitro studies with rat liver preparations have shown that 8,9-dihydro-8,9-dihydroxybenzo[k]fluoranthene is the major metabolite of benzo[k]-fluoranthene (LaVoie et al., 1980; Hecht et al., 1980).

5. EFFECTS

5.1. CARCINOGENICITY

Studies evaluating the tumorigenic potential of orally-administered benzo[k]fluoranthene could not be located in the available literature as cited in the Appendix.

Mixtures of heated beeswax and trioctanoin (1:1) containing 0.16, 0.83 or 4.15 mg benzo[k]fluoranthene (99.5% pure) were injected into the left lung lobes of 35, 31 and 27 female Osborne-Mendel rats, respectively, after thoracotomy (Deutsch-Wenzel et al., 1983). Additional groups of 35 rats served as vehicle, untreated and positive (0.1, 0.3 or 1.0 mg benzo[a]pyrene controls. Necropsies were performed on all rats at the time of natural death or when moribund, but histological examinations were limited to the lungs and organs showing gross abnormalities. Median survival times were 114, 95, 98, 104 and 118 weeks in the low-dose, middle-dose, high-dose, vehicle and untreated control groups, respectively. Median survival times for positive controls were 111, 77 and 54 weeks at its low, medium and high dose benzo[a]pyrene respectively. Squamous cell carcinomas of the lung occurred in the benzo[k]fluoranthene-treated mice at incidences of 0/35 (low dose), 3/31 (middle dose) and 12/27 (high dose). Lung tumors were not observed in either the vehicle or untreated control groups but did occur (carcinomas) at dose-related incidences in the positive controls (4/35, 21/35, 33/35). The dose-related pulmonary tumor response in the benzo[k]fluoranthene-treated mice was considered to be a treatment-related effect.

The carcinogenicity of benzo[k]fluoranthene was also evaluated in dermal studies with mice involving 2 or 3 times weekly applications for life or 13 months (Wynder and Hoffman, 1959; Habis et al., 1980), in mouse-skin initiation-promotion assays using TPA as a promoter (LaVoie et al., 1982; Amin et

al., 1985) and in a subcutaneous injection study in which mice were given three injections at monthly intervals (Lacassagne et al., 1963). As detailed in Table 5-1, benzo[k]fluoranthene was active as an initiator in the initiation-promotion assays and produced injection site sarcomas in the subcutaneous study. Interpretation of the subcutaneous injection study is complicated, however, by the lack of vehicle or untreated controls and by an unspecified observation period.

5.2. MUTAGENICITY

Benzo[k]fluoranthene was reported to induce mutations in Salmonella typhimurium strains TA100 (LaVoie et al., 1980; Amin et al., 1985) and TA98 (Hermann et al., 1980) when assayed in the presence of rat liver S-9 metabolic activation preparations (Table 5-2). The chemical was not tested in the absence of S-9.

Additional genotoxicity data for benzo[k]fluoranthene could not be located in the available literature as cited in the Appendix.

5.3. TERATOGENICITY

Pertinent data regarding the teratogenicity of benzo[k]fluoranthene could not be located in the available literature as cited in the Appendix.

5.4. OTHER REPRODUCTIVE EFFECTS

Pertinent data regarding other reproductive effects of benzo[k]fluoranthene could not be located in the available literature as cited in the Appendix.

5.5. CHRONIC AND SUBCHRONIC TOXICITY

Pertinent data regarding the of benzo[k]fluoranthene could not be located in the available literature as cited in the Appendix.

TABLE 5-1

Dermal Injection Carcinogenicity Studies of Benzo[k]fluoranthene

Route	Species/Strain	No./Sex*	Purity	Treatment	Duration	Effects/Comments	Reference
Dermal	mouse/swiss	20/F	purified	0.1 or 0.5% in acetone 3 times weekly for 13 months (volume applied not reported)	13 months	Skin papillomas developed in 0/20 (low dose) and 3/20 (high dose); no vehicle controls; terminal survival was 8/20 (low dose) and 3/20 (high dose); skin papillomas and carcinomas in 75-95% of mice similarly treated with 0.05 or 0.5% B[a]P.	Wynder and Hoffman, 1959
	mouse/NMRI	40/F	>96%	3.4, 5.6 or 9.2 µg in acetone twice weekly for life	~128 weeks	No skin tumors found in treated or vehicle control groups except one of unspecified type at the low dose survival was 75-85% after 2 years in treated groups; skin tumors in 24-69% of mice similarly treated with 1.7, 2.8 or 4.6 µg B[a]P.	Habs et al., 1980
	mouse/Crl:-1 (ICR)BR	20/F	>99%	3, 10 or 100 µg in acetone every 2 days for 20 days, followed 10 days later by 2.5 µg TPA 3 times weekly for 20 weeks	170 days (end of treatment period)	Percentages of mice with skin tumors (predominantly squamous cell papillomas) were 0, 5, 25, 75 and 85 in vehicle control, low-dose, middle-dose, high-dose and positive control (3 µg B[a]P) groups, respectively; numbers of skin tumors/mouse also dose-related.	LaVoie et al., 1982
	mouse/Crl:CD-1 (ICR)BR	20/F	>99%	101 µg in acetone every 2 days for 20 days, followed 10 days later by 2.5 µg TPA 3 times weekly for 20 weeks	170 days (end of treatment period)	Unspecified skin tumors occurred in 0 and 37% of vehicle control and treated mice, respectively; no positive control group; discrepancy in report indicates that promotion duration may have been 30 weeks.	Amin et al., 1985
Subcutaneous	mouse/XVII nc/Z	16/M, 14/F	NR	0.6 mg in olive oil once a month for 3 months	NR	Subcutaneous sarcoma in 8/16 males and 5/14 females; vehicle-treated or untreated controls not used; 16/16 males and 11/14 females alive when first tumor detected; average latency was 203 days (males) and 210 days (females); injection site tumors in 54/154 (male) and 112/162 (female) historical controls	Lacassagne et al., 1963

*Number untreated and control (if used) groups unless specified otherwise

NR - Not reported

08/06/87 0865sp

TABLE 5-2

Mutagenicity Testing of Benzo[k]fluoranthene

Assay	Indicator/ Organism	Compound/ Purity	Application	Concentration or Dose	Activating System	Response/ Comment	Reference
Reverse mutation	S. typhimurium TA100	HPLC purified	liquid suspension	10-100 µg/plate	Aroclor 1254-induced rat liver S-9	mutagenic dose-related response	LaVoie et al., 1980
	S. typhimurium TA100	>99%	NR	0.031-0.50 µmol/plate	Aroclor 1254-induced rat liver S-9	mutagenic dose-related response at doses ≥0.063 µmol/plate	Amin et al., 1985
	S. typhimurium TA98	NR	NR	5 µg/plate	rat liver S-9	mutagenic	Hermann et al., 1980

NR = Not reported

5.6. OTHER RELEVANT INFORMATION

Daily intraperitoneal injections of 0.05-40 mg/kg benzo[k]fluoranthene in arachis oil for 3 days produced dose-related increases in liver aryl hydrocarbon monooxygenase activity in rats (Schmoldt et al., 1981).

5.7. SUMMARY

Single intrapulmonary injections of 0.16, 0.83 or 4.15 mg benzo[k]fluoranthene (99.5% pure) in beeswax trioctanoin mixture into groups of 27-35 rats produced dose-related squamous cell carcinomas of the lung after lifetime observation (Deutsch-Wenzel et al., 1983). Tumors were not observed in groups of 35 vehicle or untreated controls, and incidences in the low-, middle- and high-dose treated groups were 0/35, 3/31 and 12/27, respectively.

The carcinogenicity of benzo[k]fluoranthene has also been evaluated in dermal studies with mice involving 2 or 3 times weekly applications for life or 13 months (Wynder and Hoffman, 1959; Habis et al., 1980), in mouse-skin initiation-promotion assays using TPA as a promoter (LaVoie et al., 1982; Amin et al., 1985), and in a subcutaneous injection study in which mice were given three injections at monthly intervals (Lacassagne et al., 1963). Benzo[k]fluoranthene was active as an initiator in the initiation-promotion assays and produced injection site sarcomas in the subcutaneous study. Interpretation of the subcutaneous injection study is complicated, however, by the lack of vehicle or untreated controls and by an unspecified observation period.

Benzo[k]fluoranthene induced mutations in Salmonella typhimurium strains TA100 and TA98 in the presence of exogenous metabolic activation preparations (LaVoie et al., 1980; Hermann et al., 1980; Amin et al., 1985).

Information regarding chronic or subchronic toxic effects, teratogenicity or other reproductive effects of benzo[k]fluoranthene could not be located in the available literature as cited in the Appendix.

6. AQUATIC TOXICITY

6.1. ACUTE

Pertinent data regarding the acute toxicity of benzo[k]fluoranthene to aquatic organisms could not be located in the available literature as cited in the Appendix.

6.2. CHRONIC

Pertinent data regarding the chronic toxicity of benzo[k]fluoranthene to aquatic organisms could not be located in the available literature as cited in the Appendix.

6.3. PLANTS

Pertinent data regarding the effects of benzo[k]fluoranthene on aquatic plants could not be located in the available literature as cited in the Appendix.

6.4. RESIDUES

The only information about benzo[k]fluoranthene and aquatic organisms consisted of residue monitoring data for various species and locations. Table 6-1 contains data from the study by Knutzen and Sortland (1982), who examined marine species from polluted areas in Norway; the highest concentrations were reported in mussels, Mytilus edulis (6-69 ng/kg). Tsuji et al. (1985) reported that clams (unspecified species) collected in Japanese waters contained 0.12-0.92 ng/g (presumably wet weight) benzo[k]fluoranthene. Maccubbin et al. (1985) found that the stomach contents of white suckers, Catostomus commersoni, from eastern Lake Erie contained benzo[k]fluoranthene at concentrations of 2-11 ng/g (wet weight). Dunn and Fee (1979) found that lobsters, Homarus americanus, caught in eastern Canada had an average concentration of 0.35 ng/g wet weight; however, after impoundment, the body burden was 169 ng/g.

TABLE 6-1

Monitoring Data for Benzo[k]fluoranthene in Marine Species from Norwegian Waters*

Species	Tissue Concentration (µg/kg dry weight)
INVERTEBRATE	
Mussel, *Mytilus edulis*	6-69
Periwinkle, *Littorina littorea*	3-20
Limpet, *Patella vulgata*	trace-39
Sponge, *Halichondria panicea*	40
PLANTS	
Bladder wrack, *Fucus vesiculosus*	trace-66
Knotted wrack, *Ascophyllum nodosum*	trace
Toothed wrack, *Fucus serratus*	4-15
Laminaria saccharina	4
Ceramium rubrum	6

*Source: Knutzen and Sortland, 1982

It is a common practice to store lobsters for some time before sending them to market, and frequently the enclosures are made with creosote-treated wood, a possible source of benzo[k]fluoranthene and other PAH. The 500-fold difference in benzo[k]fluoranthene levels between freshly caught and impounded lobsters indicate that the impoundment procedure may result in a significant increase in human exposure to benzo[k]fluoranthene.

6.5. SUMMARY

Data concerning the toxicity of benzo[k]fluoranthene to aquatic organisms could not be located in the available literature as cited in the Appendix. The only information consisted of monitoring data for various species and locations. Residues in species that are commonly eaten by humans were mussels in Norway, 6-69 ng/g (Knutzen and Sortland, 1982), clams in Japan, 0.12-0.92 ng/g (Tsuji et al., 1985), and lobster from Eastern Canada, 0.35 ng/g before impoundment and 169 ng/g after impoundment (Dunn and Fee, 1979).

7. EXISTING GUIDELINES AND STANDARDS

7.1. HUMAN

Exposure criteria and TLVs have been developed for PAH as a class, as well as for several individual PAH. OSHA (1985) set an 8-hour TWA concentration limit of 0.2 mg/m³ for the benzene-soluble fraction of coal tar pitch volatiles (anthracene, benzo[a]pyrene, phenanthrene, acridine, chrysene, pyrene). NIOSH (1977) recommended a concentration limit for coal tar, coal tar pitch, creosote and mixtures of these substances of 0.1 mg/m³ of the cyclohexane-extractable fraction of the sample, determined as a 10-hour TWA. NIOSH (1977) concluded that these specific coal tar products, as well as coke oven emissions, are carcinogenic and can increase the risk of lung and skin cancer in workers. NIOSH (1977) also recommended a ceiling limit for exposure to asphalt fumes of 5 mg airborne particulates/m³ of air.

Ambient water quality criteria, which specify concentration limits intended to protect humans against adverse health effects, have been recommended for PAH. U.S. EPA (1980a) recommended a concentration limit of 28 ng/ℓ for the sum of all carcinogenic PAH in ambient water. This value is based on a mathematical extrapolation of the results from studies with mice treated orally with benzo[a]pyrene and acknowledges the conservative assumption that all carcinogenic PAH are equal in potency to benzo[a]pyrene. On the basis of the animal bioassay data, daily consumption of water containing 28 ng/ℓ of carginogenic PAH over an entire lifetime is estimated to keep the lifetime risk of cancer development <1/100,000 chances.

The EPA has not recommended an ambient water quality criterion for noncarcinogenic PAH as a class. U.S. EPA (1980a) acknowledged that data suitable for quantitative risk assessment of noncarcinogenic PAH are essentially nonexistent.

7.2. AQUATIC

Guidelines and standards for the protection of aquatic biota from the effects of benzo[k]fluoranthene in particular could not be located in the available literature as cited in the Appendix. U.S. EPA (1980a) noted, however, that acute toxicity to saltwater aquatic life occurred at concentrations of 300 µg/ℓ PAH in general and would occur at lower concentrations in species more sensitive than those tested. U.S. EPA (1980a) concluded that the database was inadequate to make generalizations or recommend criteria regarding chronic toxicity or acute toxicity to freshwater biota of PAH.

8. RISK ASSESSMENT

Single intrapulmonary injections of 0.16, 0.83 or 4.15 mg benzo[k]-fluoranthene (99.5% pure) in beeswax-trioctanoin mixture into groups of 27-35 rats produced dose-related squamous cell carcinomas of the lung after lifetime observation (Deutsch-Wenzel et al., 1983). Tumors were not observed in groups of 35 vehicle or untreated controls, and incidences in the low-, middle- and high-dose treated groups were 0/35, 3/31 and 12/27, respectively. Incidences in the positive control were 4/35, 21/35 and 33/35 at 0.1, 0.3 and 1.0 mg of benzo(a)pyrene, respectively.

The carcinogenicity of benzo[k]fluoranthene was also evaluated in dermal studies with mice involving 2 or 3 times weekly applications for life or 13 months (Wynder and Hoffman, 1959; Habis et al., 1980); in mouse-skin initiation-promotion assays using TPA as a promoter (LaVoie et al., 1982; Amin et al., 1985) and in a subcutaneous injection study in which mice were given three injections at monthly intervals (Lacassagne et al., 1963). As detailed in Table 5-1, benzo[k]fluoranthene was active as an initiator in the initiation-promotion assays and produced injection site sarcomas in the subcutaneous study. Interpretation of the subcutaneous injection study is complicated, however, by the lack of vehicle or untreated controls and by an unspecified observation period.

Benzo[k]fluoranthene induced mutations in Salmonella typhimurium strains TA100 and TA98 in the presence of exogenous metabolic activation preparations (LaVoie et al., 1980; Hermann et al., 1980; Amin et al., 1985).

The results of the above studies provide sufficient evidence to conclude that benzo[k]fluoranthene is carcinogenic to experimental animals. Calculation of a carcinogenic potency factor (q_1^*) specifically for benzo[k]-fluoranthene is precluded, however, by the lack of appropriate oral or inhalation studies.

9. REPORTABLE QUANTITIES

9.1. REPORTABLE QUANTITY (RQ) RANKING BASED ON CHRONIC TOXICITY

Information regarding chronic or subchronic toxic effects, teratogencity or other reproductive effects of benzo[k]fluoranthene could not be located in the available literature as cited in the Appendix. Calculation of an RQ ranking for benzo[k]fluoranthene based on chronic toxicity is therefore precluded by the lack of appropriate data (Table 9-1).

9.2. WEIGHT OF EVIDENCE AND POTENCY FACTOR ($F=1/ED_{10}$) FOR CARCINOGENICITY

Single intrapulmonary injections of 0.16, 0.83 or 4.15 mg benzo[k]fluoranthene (99.5% pure) in beeswax-trioctanoin mixture into groups of 27-35 rats produced dose-related squamous cell carcinomas of the lung after lifetime observation (Deutsch-Wenzel et al., 1983). Tumors were not observed in groups of 35 vehicle or untreated controls, and incidences in the low-, middle- and high-dose treated groups were 0/35, 3/31 and 12/27, respectively.

The carcinogenicity of benzo[k]fluoranthene has also been evaluated in dermal studies with mice involving 2 or 3 times weekly applications for life or 13 months (Wynder and Hoffman, 1959; Habis et al., 1980), in mouse-skin initiation promotion assays using TPA as a promoter (LaVoie et al., 1982; Amin et al., 1985) and in a subcutaneous injection study in which mice were given three injections at monthly intervals (Lacassagne et al., 1963). As detailed in Table 5-1, benzo[k]fluoranthene was active as an initiator in the initiation-promotion assays and produced injection site sarcomas in the subcutaneous study. Interpretation of the subcutaneous injection study is complicated, however, by the lack of vehicle or untreated controls and by an unspecified observation period.

Benzo[k]fluoranthene induced mutations in Salmonella typhimurium strains TA100 and TA98 in the presence of exogenous metabolic activation preparations (LaVoie et al., 1980; Hermann et al., 1980; Amin et al., 1985).

TABLE 9-1

Benzo[k]fluoranthene

Minimum Effective Dose (MED) and Reportable Quantity (RQ)

Route:

Dose:

Effect:

Reference:

RV_d:

RV_e:

Composite Score:

RQ: Data are not sufficient for deriving an RQ

The results of the above studies provided sufficient evidence to conclude that benzo[k]fluoranthene is carcinogenic to experimental animals. Calculation of a carcinogenic potency factor (F) specifically for benzo[k]-fluoranthene is precluded, however, by the lack of appropriate oral or inhalation studies.

IARC (1983) has judged benzo[k]fluoranthene to be probably carcinogenic to humans (Group 2B). The corresponding EPA classification would be Group B2 (U.S. EPA, 1986c).

10. REFERENCES

Amin, S., H. Nalband, G. Balanika, K. Hule and S. Hecht. 1985. Mutagenicity and tumor initiating activity of methylated benzo[k]fluoranthenes. Cancer Lett. 26: 343-347.

Behymer, T.D. and R.A. Hites. 1985. Photolysis of polyaromatic hydrocarbons adsorbed on simulated atmospheric particles. Environ. Sci. Technol. 19: 1004-1006.

Bossert, I., W.M. Kachel and R. Bartha. 1984. Fate of hydrocarbons during oily sludge disposal in soil. Appl. Environ. Microbiol. 47: 763-767.

Cautreels, W. and K. Van Cauwenberghe. 1978. Experiments on the distribution of organic pollutants between airborne particulate matter and the corresponding gas phase. Atmos. Environ. 12: 1133-1141.

Cole, R.H., R.E. Frederick, R.P. Healy and R.G. Rolan. 1984. Preliminary findings of the priority pollutant monitoring project of the nationwide urban runoff program. J. Water Pollut. Control Fed. 56: 898-908.

Dennis, M.J., R.C. Massey, D.J. McWeeney, M.E. Knowles and D. Watson. 1983. Analysis of polycyclic aromatic hydrocarbons in UK total diets. Food Chem. Toxicol. 21: 569-574.

Deutsch-Wenzel, R.P., H. Brune, G. Grimmer, G. Dettbarn and J. Misfield. 1983. Experimental studies in rat lungs on the carcinogenicity and dose-response relationships of eight frequently occurring environmental polycyclic aromatic hydrocarbons. J. Natl. Cancer Inst. 71: 539-544.

Dunn, B.P and J. Fee. 1979. Polycyclic aromatic hydrocarbon carcinogens in commercial seafoods. J. Fish Res. Board Can. 36(12): 1469-1476.

Eiceman, G.A., R.E. Clement and F.W. Karasek. 1981. Variations in concentrations of organic compounds including PCDDS and polynuclear aromatic hydrocarbons in fly ash from a municipal incinerator. Anal. Chem. 53: 955-959.

Fazio, T. and J.W. Howard. 1983. Polycyclic aromatic hydrocarbons in foods. In: Handbook of Polycyclic Aromatic Hydrocarbons, A. Bjorseth, Ed. Marcel Dekker Inc., New York, NY. p. 461-505.

Fochtman, E.G. 1981. Biodegradation and carbon adsorption of carcinogenic and hazardous organic compounds. U.S. EPA, Cincinnati, OH. EPA 600/S2-81-032. p. 38.

Gordon, B.J. and R.J. Bryan. 1973. Patterns in airborne polynuclear hydrocarbon concentrations at four Los Angeles sites. Environ. Sci. Technol. 7: 1050-1053.

Great Lakes Water Quality Board. 1983. An inventory of chemical substances identified in the Great Lakes Ecosystem Volume 1 - Summary. Report to the Great Lakes Water Quality Board, Windsor, Ontario, Canada. p. 195.

Greenberg, A., F. Darack, R. Harkov, P. Lioy and J. Daisey. 1985. Polycyclic aromatic hydrocarbons in New Jersey: A comparison of winter and summer concentrations over a 2-year period. Atmos. Environ. 19: 1325-1339.

Griest, W.H. 1980. Multicomponent polycyclic aromatic hydrocarbon analysis of inland water and sediment. In: Hydrocarbon Halogen. Hydrocarbon Aquatic Environ., B.K. Afghan and D. Mackay, Ed. Plenum Press, New York, NY. p. 173-183.

Habis, M., D. Schmaehl and J. Misfeld. 1980. Local carcinogenicity of some environmentally relevant polycyclic aromatic hydrocarbons after lifelong topical application to mouse skin. Arch. Geschwulstforsch. 50(3): 266-274.

Harkov, R., A. Greenberg, F. Darack, J.M. Daisey and P.J. Lioy. 1984. Summertime variations in polycyclic aromatic hydrocarbons at four sites in New Jersey. Environ. Sci. Technol. 18: 287-291.

Hecht, S.S, E. LaVoie, S. Amin, V. Bedenko and D. Hoffman. 1980. A study of chemical carcinogenesis. Part 23. On the metabolic activation of the benzofluoranthenes. In: Polynuclear Aromatic Hydrocarbons: Chemistry and Biological Effects, Int. Symp., 1979. Battelle Press, Columbus, OH. p. 417-433.

Heit, M. 1985. The relationship of a coal fired power plant to the levels of polycyclic aromatic hydrocarbons (PAH) in the sediment of Cayuga Lake. Water Air Soil Pollut. 24: 41-61.

Hermann, M., J.P. Durand, J.M. Charpentier, et al. 1980. Correlations of mutagenic activity with polynuclear aromatic hydrocarbon content of various mineral oils. In: Polynuclear Aromatic Hydrocarbons: Chemistry and Biological Effect, 4th Int. Symp., A. Bjorseth and A.J. Dennis, Ed. Battelle Press, Columbus, OH. p. 899-916. (Cited in IARC, 1983)

IARC (International Agency for Research on Cancer). 1983. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Benzo[k]-fluoranthene. In: Polynuclear Aromatic Compounds, Part 1, Chemical, Environmental and Experimental Data. WHO, IARC, Lyons, France. Vol. 32, p. 163-170.

Johnson, A.C., P.F. Larsen, D.F. Gadbois and A.W. Humason. 1985. The distribution of polycyclic aromatic hydrocarbons in the surficial sediments of Penobscot Bay (Maine, USA) in relation to possible sources and other sites world wide. Marine Environ. Res. 15: 1-16.

Keller, C.D. and T.F. Bidleman. 1984. Collection of airborne polycyclic aromatic hydrocarbons and other organics with a glass fiber filter-polyurethane foam system. Atmos. Environ. 18: 837-845.

Knutzen, J. and B. Sortland. 1982. Polycyclic aromatic hydrocarbons (PAH) in some algae and invertebrates from moderately polluted parts of the coast of Norway. Water Res. 16(4): 421-428.

Kotin, P., H.L. Falk and R. Busser. 1969. Distribution, retention and elimination of C^{14}-3,4-benzpyrene after administration to mice and rats. J. Natl. Cancer Inst. 23: 541.

Kveseth, K., B. Sortland and T. Bokn. 1982. Polycyclic aromatic hydrocarbons in sewage, mussels and tap water. Chemosphere. 11: 623-639.

Lacassagne, A., N.P. Buu-Hoi, F. Zajdela, D. Lavit-Lamy and O. Chalvet. 1963. Carcinogenic activity of polycyclic aromatic hydrocarbons with a fluoranthene group. Unio Int. Contra Cancrum Acta. 19:490-496. (Cited in IARC, 1983)

Lane, D.A. and M. Katz. 1977. The photomodification of benzo(a)pyrene, benzo(b)fluoranthene and benzo(k)fluoranthene under simulated atmospheric conditions. Adv. Environ. Sci. Technol. 8(Fate Pollut Air Water Environ.): 137-154.

LaVoie, E.J., S.S. Hecht, S. Amin, V. Bedenko and D. Hoffman. 1980. Identification of mutagenic dihydrodiols as metabolites of benzo[j]fluoranthene and benzo[k]fluoranthene. Cancer Res. 40: 4528-4532.

LaVoie, E.J, S. Amin, S.S. Hecht, K. Furuya and D. Hoffman. 1982. Tumor initiating activity of dihydrodiols of benzo[b]fluoranthene, benzo[j]fluoranthene and benzo[k]fluoranthene. Carcinogenesis (London). 3(1): 49-52.

Ligocki, M.P., C. Leuenberger and J.F. Pankow. 1985a. Trace organic compounds in rain-II. Gas scavenging of neutral organic compounds. Atmos. Environ. 19: 1609-1619.

Ligocki, M.P., C. Leuenberger and J.F. Pankow. 1985b. Trace organic compounds in rain-III. Particle scavenging of neutral organic compounds. Atmos. Environ. 19: 1619-1626.

Lunde, G. and A. Bjoerseth. 1977. Polycyclic aromatic hydrocarbons in long-range transported aerosols. Nature. 268: 518-519.

Lyman, W.J., W.F. Reehl and D.H. Rosenblatt. 1982. Handbook of Chemical Property Estimation Methods. McGraw-Hill Book Co., New York. p. 15-16, 15-29, 4-9, 5-4, 5-10.

Mabey, W.R., J.H. Smith, R.T. Podoll, et al. 1981. Aquatic fate process data for organic priority pollutants. U.S. EPA, Monitoring and Data Support Division (WH 553), Office of Water Regulations and Standards, Washington, DC. EPA 440/4-81-014.

Maccubbin, A.E., P. Black, L. Trzeciak and J.J. Black. 1985. Evidence for polynuclear aromatic hydrocarbons in the diet of bottom-feeding fish. Bull. Environ. Contam. Toxicol. 34(6): 876-882.

Marty, J.C., M.J. Tissier and A. Saliot. 1984. Gaseous and particulate polycyclic aromatic hydrocarbons (PAH) from the marine atmosphere. Atmos. Environ. 18: 2183-2190.

Matsumoto, H. and T. Kashimoto. 1985. Average daily respiratory intake of polycyclic aromatic hydrocarbons in ambient air determined by capillary gas chromatography. Bull. Environ. Contam. Toxicol. 34: 17-23.

McFall, J.A., S.R. Antoine and I.E. Deleon. 1985. Base-neutral extractable organic pollutants in biota and sediments from Lake Pontchartrain. Chemosphere. 14: 1561-1569.

Muel, B. and S. Saguem. 1985. Determination of 23 polycyclic aromatic hydrocarbons in atmospheric particulate matter of the Paris area and photolysis by sunlight. Int. J. Environ. Anal. Chem. 19: 111-131.

NAS (National Academy of Sciences). 1972. Biologic Effects of Atmospheric Pollutants. Particulate Polycyclic Organic Matter. NAS, Washington, DC. p. 63-81.

NIOSH (National Institute for Occupational Safety and Health). 1977. Criteria for a Recommended Standard...Occupational Exposure to Coal Tar Products. U.S. DHEW, PHS, CDC, Rockville, MD. Publ. No. 78-107.

NRC (National Research Council). 1983. Polycyclic Aromatic Hydrocarbons: Evaluation of Sources and Effects. NRC, National Academy Press, Washington, DC.

OSHA (Occupational Safety and Health Administration). 1985. OSHA Safety and Health Standards. 29 CFR 1910.1000.

Pearlman, R.S., S.H. Yalkowski and S. Banerjee. 1984. Water solubilities of polynuclear aromatic and heteroaromatic compounds. J. Chem. Ref. Data. 13: 555-562.

Prahl, F.G., E. Crecelius and R. Carpenter. 1984. Polycyclic aromatic hydrocarbons in Washington coastal sediments: An evaluation of atmospheric and riverene roots of introduction. Environ. Sci. Technol. 18: 687-693.

Readman, J.W., R.F.C. Mantoura, M.M. Rhead and L. Brown. 1982. Aquatic distribution and heterotrophic degradation of polycyclic aromatic hydrocarbons (PAH) in the Tamar Estuary. Estuarine, Coastal. Shelf. Sci. 14: 369-389.

Rees, E.D., P. Mandelstam, J.Q. Lowry and H. Lipscomb. 1971. A study of the mechanism of intestinal absorption of benzo[a]pyrene. Biochem. Biophys. Acta. 225: 96.

Rostad, C.E., W.A. Pereiara and M.F. Hult. 1985. Partitioning studies of coal-tar constituents in a two-phase contaminated groundwater system. Chemosphere. 14: 1023-1036.

Ruepert, C., A. Grinwis and H. Govers. 1985. Prediction of partition coefficients of unsubstituted polycyclic aromatic hydrocarbons from C_{18} chromatographic and structural properties. Chemosphere. 14: 279-291.

Santodonato, J., P. Howard and D. Basu. 1981. Health and Ecological Assessment of Polynuclear Aromatic Hydrocarbons. Pathotox Publishers, Inc., Park Forest South, IL.

Saxena, J., D.K. Basu and J. Kozuchowski. 1977. Method development and monitoring of polynuclear aromatic hydrocarbons in selected U.S. waters. EPA 600-11-77-052. U.S. EPA, Cincinnati, OH. p. 84.

Schmoldt, A., J. Jacob and G. Grimmer. 1981. Dose-dependent induction of rat liver microsomal aryl hydrocarbon monooxygenase by benzo[k]fluoranthene. Cancer Lett. 13(3): 249-257.

Shackelford, W.M. and L.H. Keith. 1976. Frequency of Organic Compounds Identified in Water. EPA 600/4-76-062. p. 79.

Sims, R.C. and M.R. Overcash. 1983. Fate of polynuclear aromatic compounds (PNAS) in soil-plant systems. Residue Rev. 88: 1-68.

Sorrell, R.K., H.J. Brass and R. Reding. 1980. A review of the occurrences and treatment of polynuclear aromatic hydrocarbons in water. Environ. Int. 4: 245-254.

Staples, C.A., A. Werner and T. Hoogheem. 1985. Assessment of priority pollutant concentrations in the United States using STORET database. Environ. Toxicol. Chem. 4: 131-142.

Tan, Y.L. and M. Heit. 1981. Biogenic and abiogenic polynuclear aromatic hydrocarbons in sediments from two remote Adirondack lakes. Geochim Cosmochim. Acta. 45: 2267-2279.

Tsuji, I., K. Koide, H. Moriyama, K. Tanabe and H. Matsushita. 1985. Detection of polynuclear aromatic hydrocarbons in shellfishes, seabottom sediments and sea water by high performance liquid chromatography. Shokuhin Eiseigaku Zasshi. 26(1): 50-55.

U.S. EPA. 1980a. Ambient Water Quality Criteria Document for Polynuclear Aromatic Hydrocarbons. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Water Regulations and Standards, Washington, DC. EPA 440/5-80-069. NTIS PB-81-117866.

U.S. EPA. 1980b. Guidelines and Methodology Used in the Preparation of Health Effects Assessment Chapters of the Consent Decree Water Criteria Documents. Federal Register. 45(231): 49347-49357.

U.S. EPA. 1982. An Exposure and Risk Assessment for Benzo[a]pyrene and Other Polycyclic Aromatic Hydrocarbons. Prepared by the Office of Water Regulations and Standards, Washington, DC. Final Draft Report.

U.S. EPA. 1984. Health Effects Assessment for Polycyclic Aromatic Hydrocarbons (PAH). Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Emergency and Remedial Response, Washington, DC.

U.S. EPA. 1986a. OHM-TADS (Oil and Hazardous Materials Technical Assistance Data System). On-line: April 16, 1986.

U.S. EPA. 1986b. Graphical Exposure Modeling System (GEMS). Octanol-Water Partition Coefficient (CLOGP) and Fate of Atmospheric Pollutants (FAP) data bases. Computer Estimations, August 8, 1986. U.S. EPA, Washington, DC.

U.S. EPA. 1986c. Guidelines for Carcinogen Risk Assessment. Federal Register. 51(185): 33992-34003.

USITC (U.S. International Trade Commission). 1984. Imports of Benzenoid Chemicals and Products, 1983. USITC Publ. 1548, Washington, DC.

Vaessen, H.A.M.G., P.L. Schuliter, A.A. Jekel and A.A.M.M. Wilbers. 1984. Polycyclic aromatic hydrocarbons in selected foods; analysis and occurrence. Toxicol. Environ. Chem. 7: 297-324.

Vainio, H., P. Volila, J. Hartiola and O. Pelkonen. 1976. The fate of intratracheally installed benzo[a]pyrene in the isolated perfused rat lung of both control and 20-methylcholanthrene pretreated rats. Res. Comm. Chem. Pathol. Pharmacol. 13: 259-271. (Cited in U.S. EPA, 1980a)

Van Noort, P.C.M. and E. Wondergem. 1985. Scavenging of airborne polycyclic aromatic hydrocarbons by rain. Environ. Sci. Technol. 19: 1044-1048.

Weast, R.C., Ed. 1980. CRC Handbook of Chemistry and Physics, 61st ed. CRC Press Inc., Boca Raton, FL. p. 155.

Wynder, E.L. and D. Hoffman. 1959. The carcinogenicity of benzofluoranthenes. Cancer. 12: 1194-1199.

Yamasaki, H., K. Kuwata and Y. Kuge. 1985. Effects of vapor pressure of polycyclic aromatic hydrocarbons on their adsorption/description between vapor phase and particulate phase in ambient air. Nippon Kagaku Kaishi. 8: 1632-1624.

Zoeteman, B.C.J., E. Degreef and F.J.J. Brinkman. 1981. Persistence of organic contaminants in ground water, lessons from soil pollution incidents in the Netherlands. Sci. Total Environ. 21: 187-202.

APPENDIX

LITERATURE SEARCHED

This profile is based on data identified by computerized literature searches of the following:

GLOBAL
TSCATS
CASR online (U.S. EPA Chemical Activities Status Report)
CAS online STN International
TOXLINE
TOXBACK 76
TOXBACK 65
RTECS
OHM TADS
STORET
SRC Environmental Fate Data Bases
SANSS
AQUIRE
TSCAPP
NTIS
Federal Register

These searches were conducted in April, 1986. In addition, hand searches were made of Chemical Abstracts (Collective Indices 6 and 7), and the following secondary sources were reviewed:

ACGIH (American Conference of Governmental Industrial Hygienists). 1986. Documentation of the Threshold Limit Values and Biological Exposure Indices, 5th ed. Cincinnati, OH.

ACGIH (American Conference of Governmental Industrial Hygienists). 1985-1986. TLVs: Threshold Limit Values for Chemical Substances and Physical Agents in the Workroom Environment with Intended Changes for 1985-1986. Cincinnati, OH. 114 p.

Clayton, G.D. and F.E. Clayton, Ed. 1981. Patty's Industrial Hygiene and Toxicology, 3rd rev. ed., Vol. 2A. John Wiley and Sons, NY. 2878 p.

Clayton, G.D. and F.E. Clayton, Ed. 1981. Patty's Industrial Hygiene and Toxicology, 3rd rev. ed., Vol. 2B. John Wiley and Sons, NY. p. 2879-3816.

Clayton, G.D. and F.E. Clayton, Ed. 1982. Patty's Industrial Hygiene and Toxicology, 3rd rev. ed., Vol. 2C. John Wiley and Sons, NY. p. 3817-5112.

Grayson, M. and D. Eckroth, Ed. 1978-1983. Kirk-Othmer Encyclopedia of Chemical Technology, 3rd ed. John Wiley and Sons, NY. 23 Volumes.

Hamilton, A. and H.L. Hardy. 1974. Industrial Toxicology, 3rd ed. Publishing Sciences Group, Inc., Littleton, MA. 575 p.

IARC (International Agency for Research on Cancer). IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Humans. WHO, IARC, Lyons, France.

NTP (National Toxicology Program). 1986. Toxicology Research and Testing Program. Chemicals on Standard Protocol. Management Status.

Ouellette, R.P. and J.A. King. 1977. Chemical Week Pesticide Register. McGraw-Hill Book Co., NY.

Sax, N.I. 1979. Dangerous Properties of Industrial Materials, 5th ed. Van Nostrand Reinhold Co., NY.

SRI (Stanford Research Institute). 1984. Directory of Chemical Producers. Menlo Park, CA.

U.S. EPA. 1985. Status Report on Rebuttable Presumption Against Registration (RPAR) or Special Review Process. Registration Standards and the Data Call in Programs. Office of Pesticide Programs, Washington, DC.

U.S. EPA. 1985. CSB Existing Chemical Assessment Tracking System. Name and CAS Number Ordered Indexes. Office of Toxic Substances, Washington, DC.

USITC (U.S. International Trade Commission). 1983. Synthetic Organic Chemicals. U.S. Production and Sales, 1982, USITC Publ. 1422, Washington, DC.

Verschueren, K. 1983. Handbook of Environmental Data on Organic Chemicals, 2nd ed. Van Nostrand Reinhold Co., NY.

Windholz, M., Ed. 1983. The Merck Index, 10th ed. Merck and Co., Inc., Rahway, NJ.

Worthing, C.R. and S.B. Walker, Ed. 1983. The Pesticide Manual. British Crop Protection Council. 695 p.

In addition, approximately 30 compendia of aquatic toxicity data were reviewed, including the following:

Battelle's Columbus Laboratories. 1971. Water Quality Criteria Data Book. Volume 3. Effects of Chemicals on Aquatic Life. Selected Data from the Literature through 1968. Prepared for the U.S. EPA under Contract No. 68-01-0007. Washington, DC.

Johnson, W.W. and M.T. Finley. 1980. Handbook of Acute Toxicity of Chemicals to Fish and Aquatic Invertebrates. Summaries of Toxicity Tests Conducted at Columbia National Fisheries Research Laboratory. 1965-1978. U.S. Dept. Interior, Fish and Wildlife Serv. Res. Publ. 137, Washington, DC.

McKee, J.E. and H.W. Wolf. 1963. Water Quality Criteria, 2nd ed. Prepared for the Resources Agency of California, State Water Quality Control Board. Publ. No. 3-A.

Pimental, D. 1971. Ecological Effects of Pesticides on Non-Target Species. Prepared for the U.S. EPA, Washington, DC. PB-269605.

Schneider, B.A. 1979. Toxicology Handbook. Mammalian and Aquatic Data. Book 1: Toxicology Data. Office of Pesticide Programs, U.S. EPA, Washington, DC. EPA 540/9-79-003. NTIS PB 80-196876.